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1: FEBS Lett. 1998 Oct 16;437(1-2):137-41.

Regulation of the fibronectin EDA exon alternative splicing. Cooperative role of the exonic enhancer element and the 5' splicing site.

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Alternatively spliced exons generally contain weak splicing sites, and exonic and/or intronic regulatory elements recognised by trans-acting auxiliary splicing factors. The EDA exon of the fibronectin gene is a typical example of an exon bearing a purine-rich exon splicing enhancer (ESE) element recognised by members of the SR phosphoprotein family. The regulatory region that governs splicing in the human EDA exon also contains an exon splicing silencer (ESS) element. We have cloned the mouse EDA genomic region, and we show that the ESE and the ESS elements, although they have base differences, can be replaced by the human elements without significant change in the exon inclusion/exclusion ratio. This fact suggests a common splicing regulatory mechanism across species. We demonstrate in vivo the functional activity of the mouse ESE element in splicing. We also show that the trans-acting factors recognising this element cooperate with the 5' splicing site of the EDA exon to facilitate proper exon recognition. Indeed, a strong 5' splicing site overrides the ESE function in exon recognition. However, the presence of a strong 3' splicing site is not sufficient to compensate for the absence of the splicing enhancer. Our data provide in vivo evidence of the interplay between the exonic splicing regulatory elements and the splicing sites, leading finally to subtle regulation of alternative splicing.

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